not due to CAD. Identification and reduction of risk factors is important in preventing morbidity and mortality due to CAD in females.

High versus low intensity statin therapy prior to thrombolysis in Indian patients with acute ST-segment elevation myocardial infarction

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Objectives: This study sought to compare high intensity statin versus low intensity statin therapy in Indian patients with ST-segment elevation myocardial infarction (STEMI) undergoing thrombolysis.
Background: Previous randomized trials have demonstrated that statin pre-treatment reduced major adverse cardiac events (MACEs) in patients with stable angina pectoris and acute coronary syndrome. However, randomized studies of statin therapy in Indian patients with STEMI are scarce.
Methods: Of 1230 patients with acute STEMI, 460 patients satisfied the inclusion criteria and were randomized to 80-mg atorvastatin ($n = 225$) or 10-mg atorvastatin ($n = 235$) arms for pre-treatment before thrombolytic therapy. The primary end point was 30-day incidence of MACE including death and nonfatal MI. Secondary end points included readmission and ST-segment resolution at 90 min after thrombolysis.
Results: The two groups did not differ in their primary endpoints. MACE occurred in 12 (5.33%) and 14 (5.96%) patients in the 80-mg and 10-mg atorvastatin pre-treatment arms, respectively ($p = 0.92$). But ST-segment resolution was significantly higher in the 80-mg atorvastatin arm (64.87 ± 14.84 vs $54.84 \pm 16.01\%$, $p < 0.001$). Of note, myalgia was significantly more in 80 mg statin group (18.22% vs 7.66%, $p = 0.001$).
Conclusions: High-dose atorvastatin pre-treatment before thrombolysis did not show a significant difference of MACEs compared with low dose atorvastatin but did show significant improvement in immediate coronary flow after thrombolysis as depicted by ST-segment resolution. This benefit at the cost of subjecting significantly greater number of patients to significant myalgia, questions the usefulness of high dose statin in Indian patients.

Ivabradine versus metoprolol in patients with acute inferior wall myocardial infarction – 'Expanding arena for ivabradine'

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Background: Beta blockers in ST-segment elevation myocardial infarction (STEMI) are indicated in patients with high heart rates (HR) or left ventricular (LV) dysfunction. But atrioventricular (AV) blocks are the biggest concern in inferior MI with beta blockers. In contrast ivabradine may lower heart rate with a lesser risk of AV blocks.
Aim: To investigate the feasibility, tolerability, and the efficacy of ivabradine versus metoprolol in acute inferior STEMI and during 30 days of follow up.
Methods: It was a prospective double blind single centre randomized controlled study. Of 1032 patients with acute inferior STEMI, 564 patients did not fulfill the inclusion criteria and were excluded. 468 patients were included in the study and were randomized in 1:1 manner to ivabradine (group A) and metoprolol (group B). 42 patients were lost on follow up and excluded. Per protocol analysis of 426 patients (group A – 210 and group B – 216) was done. The primary end point was 30-days incidence of MACE including death, reinfarction, complete heart block (CHB), and heart failure. Secondary endpoints included 30 days incidence of recurrent angina, readmission, first or second degree AV block, and tachyarrhythmias.
Results: Both the drugs decreased the mean heart rate to 62.22 ± 2.95 (group A) vs 62.53 ± 3.59 (group B) beats per minute ($p = 0.33$). Ejection fraction improved in both the groups (2.4% in group A vs 3.2% in group B). The two groups did not differ significantly in their primary endpoints in terms of death (group A = 1.90% vs group B = 1.85%, OR = 1.03, 95% CI = 0.25–4.17, $p = 0.97$), reinfarction (group A = 0.95% vs group B = 0.93%, OR = 1.03, 95% CI = 0.14–7.37, $p = 0.98$), heart failure (group A = 4.76% vs group B = 2.78%, OR = 1.75, 95% CI = 0.62–4.90, $p = 0.29$), or CHB (0% vs 2.78%, OR = 0.08, 95% CI = 0.004–1.37, $p = 0.08$). There were no significant differences in the secondary end points of recurrent angina, readmission, and tachyarrhythmias but significantly more first degree AV blocks occurred with metoprolol (13.89% vs 2.86%, OR = 5.48, 95% CI = 2.23–13.47, $p = 0.0002$).
Conclusions: Ivabradine is well tolerated and equally effective as metoprolol in acute inferior wall STEMI patients for lowering the heart rate with significant less risk of AV blocks.

Bifurcation stenting – A single center experience

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Bifurcation is the division of an artery into 2 branches and it is a common anatomical feature of the human coronary tree. Bifurcation lesions are recognized as a common site for atherosclerotic plaque build up and account for 15–20% of all interventions. These lesions are complex and challenging for percutaneous intervention. Numerous anatomic patterns of bifurcation stenosis are present and there is no consistent and reliable methodology to address these complex lesions, that is, there is no ''one size fits all'' solution to the bifurcation puzzle. The optimal percutaneous coronary intervention technique remains undetermined.
Method: We analysed all the bifurcation lesion stenting procedures done at our institution for three years from 2012 to 2015.
Results: Total of 138 cases of bifurcation stenting was done over a period of three years. There were 96 males and 42 females. Anterior wall myocardial infarction accounted for 89% of all cases and the remaining were inferior wall myocardial infarctions.
True bifurcation lesions were 53 in number.
86 cases had bifurcation lesion involving the LAD, 45 involved the LCX, and 7 involved the RCA.
The predominant method of stenting was ''T'' at provision (TAP) and involved 91 cases.
12 cases underwent ''Minicrush'', 8 underwent simultaneous kissing stent (SKS) technique, and 27 cases underwent Classic T stenting.
Conclusion: The predominant technique of stenting was the TAP technique, which was always followed by proximal optimization of stent (POTS) to ensure side branch patency. Most of the bifurcation

lesions can be addressed by this technique thereby reducing the cost, as a single stent is sufficient and also, the amount of metal at the bifurcation is reduced, thereby reducing the incidence of restenosis. To conclude TAP is a safe, easy, and cost effective technique that can be used to address most of the bifurcation lesions.

On treatment platelet reactivity in post percutaneous coronary intervention patients on thienopyridine drugs: A six-month outcomes study

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Background: In the present era of percutaneous coronary intervention (PCI) dual antiplatelet therapy plays a pivotal role. However there exists inter individual variability in response to antiplatelet therapy. Thus, monitoring antiplatelet therapy becomes vital and may identify poor responders who would benefit from the change in therapy.
Objectives: We sought to identify the efficacy of thienopyridine antiplatelet drugs in post PCI patients, by using AggreGuide A-100 Platelet Aggregometer, point of care device developed to monitor platelet aggregation using laser scattering technique.
Methods: This prospective, single-center study, includes patients who received thienopyridine antiplatelet drugs after PCI during May 2014–January 2015. In these patients, platelet aggregation inhibition was evaluated from the whole blood sample using AggreGuide A-100 and expressed in terms of platelet activity index – PAI. Patients were labelled as poor responders (high on treatment platelet reactivity) if the PAI was more than 5. Genetic polymorphisms of ABCB1 gene were studied in Clopidogrel group.
Results: A total of 405 patients were enrolled in this study. Among them 109 (26.9%) were female and 296 (83.1%) were males, 108 (26.7%) underwent primary PCI and 297 (83.7%) underwent elective PCI. At the discretion of treating physician 221 (54.6%), 111 (27.4%), and 73 (18%) patients received Clopidogrel, Prasugrel, and Ticagrelor, respectively. It is found that therapy was ineffective (PAI $>$ 5) in 73 (16%) patients of which 52, 8, and 5 were in Clopidogrel, Prasugrel, and Ticagrelor groups, respectively. Cumulatively, there was high PAI in females and those who underwent primary PCI. In the Clopidogrel group those with additional Cilostazol were having low PAI ($p < 0.05$). Bleeding complications were observed in 3, 9, and 3 patients of Clopidogrel, Prasugrel, and Ticagrelor groups, respectively. Four patients in Clopidogrel group had reinfarction. Antiplatelet therapy was optimised in those with high PAI.
Conclusion: It is found that there is high resistance to antiplatelet drugs (especially Clopidogrel), and thus monitoring individual's platelet reactivity and optimising therapy based on it, should become a new standard-of-care for patients on antiplatelet therapy.

Electrocardiogram: A simple tool to predict angiographic localization of coronary lesions in STEMI patients

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Background: Rapid risk stratification of the patient with acute chest pain is essential to select the best management. Although culprit lesions in STEMI cluster in the proximal coronary arteries, their relationship to bifurcations and curvatures, where blood flow is disturbed, is unknown.
Objective: We investigated the value of the ECG at first medical contact to determine location and size of the ischemic myocardial area and thereby severity of risk. We also hypothesized that (a) culprit lesions are mostly localized to areas with disturbed flow, e.g. distal to bifurcations and curvatures and (b) the distribution of culprit lesions in the left coronary artery (LCA) and right coronary artery (RCA) and resulting infarct size are related to the location of bifurcations and curvatures.
Methods: In patients with ST elevation myocardial infarction (STEMI), ECG findings were correlated with the coronary angiogram. Using ST-segment deviation patterns the location of the coronary culprit lesion was predicted and also the size of the myocardium at risk. By quantitative coronary angiography, the distances from the vessel ostium, major bifurcations, and major curvatures to the culprit lesion were measured in 104 patients.
Results: Correct coronary culprit artery identification was possible in 96% of the patients and this was most accurate in localization of culprit lesion in the proximal part of the coronary arteries. Culprit lesions were located within 20 mm of a bifurcation in 77% of patients and closer to the bifurcation in the LCA compared with the RCA (10.13 ± 7.09 vs 18.49 ± 17.16 mm, $p = 0.01$). Of RCA culprit lesions, 53% were located within 30 mm of a major curvature. Compared with those in the RCA, culprit lesions in the LCA were located more proximally (19.28 ± 10.27 vs 28.77 ± 20.65 mm, $p < 0.01$) and were associated with larger myocardial infarctions as assessed by CK-MB (139.5 ± 139.4 vs 89.2 ± 93.9 unit/L, $p = 0.03$), and lower ejection fractions ($39.58 \pm 7.0\%$ vs $45.16 \pm 8.75\%$, $p = 0.001$).
Conclusions: Admission ECG gives adequate insight regarding the infarct related artery and the location of the lesion in the culprit vessel. In patients with STEMI, culprit lesions are frequently located immediately distal to bifurcations and in proximity to major curvatures where disturbed flow is known to occur. This supports the role of wall shear stress in the pathogenesis of STEMI

Serum uric acid as a marker of coronary artery disease – A single center study

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Background: Uric acid is the end product of nucleotide metabolism. Serum uric acid when elevated is diagnostic of gout and is a stress marker in hypertension. The correlation of serum uric acid with coronary artery disease has been evaluated though in small number cohort. Present study aims to correlate the serum uric acid with the severity of CAD and as a marker of CAD.
Materials and methods: A total of 470 patients consecutively have been enrolled into the study. The patients with already a diagnosis of gout, on long term use of thiazides and those responsible for elevated serum urate levels were excluded from the study. The patients enrolled underwent coronary angiography after informed and written consent. Based on the results of CAG the patients were divided into two groups, CAD ($n = 350$) and control group ($n = 120$). The severity of CAD was assessed by modified Gensini score.
Results: Mean age of presentation in CAD group was 53.72 ± 10.38 yrs and in controls was 49.60 ± 9.82 yrs ($p < 0.0001$). Males in total cohort were 331 (70.43%). Hypertensives 226 vs 82 ($p = 0.54$), diabetics (161 vs 30, $p = 0.60$), smokers (209 vs 48, $p = 0.64$),